Diagnostic Ophthalmology
Ophtalmologie diagnostique

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History and clinical signs

A 4-year-old spayed female Cavalier King Charles spaniel dog was examined at the ophthalmology service at the Western College of Veterinary Medicine for evaluation of white spots in the corneas. The menace responses, palpebral, occulocephalic, and direct and consensual pupillary light reflexes were present bilaterally. Schirmer tear test (Schirmer Tear Test Strips; Alcon Canada, Mississauga, Ontario) values were 22 mm/min bilaterally. The intraocular pressures were estimated with a rebound tonometer (Tonvet; Tiolat, Helsinki, Finland) and were 17 and 18 mmHg in the right and left eye, respectively. Fluorescein staining (Fluorets; Bausch & Lomb Canada, Markham, Ontario) was negative bilaterally. On direct examination there were 3 mm diameter, dense, oval, refractile, white opacities in the axial corneas of both eyes. Biomicroscopic examination (Osram 64222; Carl Zeiss Canada, Don Mills, Ontario) revealed the opacities to consist of multiple fine crystalline particles located in the superficial corneal stroma. Following application of 0.5% tropicamide (Mydriacyl; Alcon Canada, Mississauga, Ontario) indirect ophthalmoscopic (Heine Omega 200; Heine Instruments Canada, Kitchener, Ontario) examinations were completed bilaterally and abnormalities were not detected. A photograph of the right eye at presentation is provided for your assessment (Figure 1).

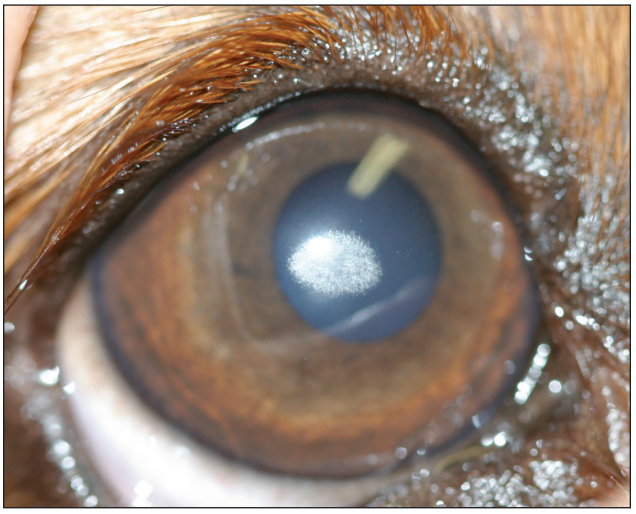

Figure 1. Photograph of the right eye of a 4-year-old Cavalier King Charles spaniel.

What are your clinical diagnosis, differential diagnoses, therapeutic plan, and prognosis?

Discussion

Our clinical diagnosis was corneal stromal dystrophy. Corneal stromal dystrophy is a primary, inherited, bilateral opacity of the cornea which is not associated with ocular inflammation or systemic disease. Opacities due to stromal dystrophy are depositions of cholesterol, lipid, and fatty acids within the stroma. Corneal stromal dystrophy occurs in many dog breeds and has been well described in the beagle, Siberian husky, Airedale terrier, rough collie, and Cavalier King Charles spaniel (1). Mode of inheritance is known for some breeds, but is variable among breeds.

The main clinical manifestation of corneal stromal dystrophy is a bilateral, roughly symmetrical, round to oval crystalline opacity in the axial or paraxial cornea that is not associated with corneal inflammation, vascularization, or pigmentation. In the Cavalier King Charles spaniel specifically, opacities develop between 2 to 4 years of age and consist of fine, striate crystals closely packed together in the sup-epithelial, axial, or paraxial cornea. The pathogenesis is unknown but speculated to be a local abnormality of corneal fibroblasts, possibly with lipid metabolism and a polygenic mode of inheritance is suspected (2).

The diagnosis of corneal stromal dystrophy is based on the signalment and clinical manifestations described. The differential diagnoses for corneal dystrophy include corneal fibrosis, corneal degeneration, and lipid keratopathies; all of which are not inherited or familial, are non-symmetrical, and not necessarily bilateral. Corneal fibrosis occurs following corneal stromal injury and is usually a well-demarcated dense gray-white opacity. Corneal degeneration occurs secondary to pathological conditions of the cornea, or systemic disease causing lipid, cholesterol, or calcium deposition. Corneal inflammation, vascularization, and/or melanosis often accompany the changes (3). Lipid keratopathy is associated with systemic lipid abnormalities and is characterized by peripheral and/or central, crystalline corneal opacities in one or both eyes (1,3). When lipid or mineral deposition is not typical of inherited dystrophy, serum chemistry and lipid profiles are recommended to investigate for underlying systemic conditions.

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In general, corneal stromal dystrophies do not require or respond to medical therapy. Topical anti-inflammatory medications may actually exacerbate the lesion (3). Lesions may return after keratectomy and therefore, surgery is not usually recommended. Low-fat diets have been recommended anecdotally; however, there are no clinical studies showing that reducing fat in the diet has any significant effect on the density or progression of the lesion.

The prognosis for corneal stromal dystrophies is excellent. Most opacities are small and do not progress to involve the whole cornea. They do not tend to cause visual disturbance and they are usually not associated with ocular pain.

References

1. Ledbetter EC, Gilger BC. Diseases and Surgery of the Canine Cornea and Sclera. In: Gelatt KN, ed. Veterinary Ophthalmology. 5th ed. Ames, Iowa: Wiley-Blackwell Publishing, 2013:976–1049.
2. Crispin S. Crystalline stromal dystrophy in the Cavalier King Charles spaniel. Proceedings of the 17th Annual Meeting of the American College of Veterinary Ophthalmologists, New Orleans, Louisiana, 1987:18–23.
3. Crispin S, Barnett K. Dystrophy, degeneration, and infiltration of the canine cornea. J Small Anim Pract 1983;24:63–83.

Answers to Quiz Corner
Les réponses du test éclair

1. **d)** Regular insulin, which is the most potent, and which can be given IV if desired, is the most appropriate treatment for this dog.

d) L'insuline ordinaire, qui est la plus puissante, et qui peut être administrée par voie intraveineuse, si désiré, est le traitement le plus approprié pour ce chien.

2. **d)** Hypertrophic cardiomyopathy is associated with concentric ventricular hypertrophy and left atrial enlargement. Maine coon cats have an increased incidence of hypertrophic cardiomyopathy compared with other cats.

d) La cardiomyopathie obstructive est associée à l'hypertrophie ventriculaire concentrique et à l'agrandissement de l'oreillette gauche. Les chats Maine coon possèdent une incidence élevée de cardiomyopathie obstructive par comparaison aux autres chats.

3. **a)** Ionophores, intentionally added to many livestock diets to promote growth and act as coccidiostats, can cause cardiac effects if the recommended dosage in the diet is exceeded. Selenium deficiency can cause cardiac effects, but these effects are not as acute as those described. Urea, copper, and ergot are not cardiotoxic.

a) Les ionophores, ajoutés intentionnellement à plusieurs diètes du bétail pour accélérer la croissance et agir comme corticostatiques, peuvent causer des dommages cardiaques, si la dose recommandée dans la diète est dépassée. Une carence en sélénium peut causer des dommages cardiaques, mais ceux-ci ne sont pas aussi aigus que ceux qui sont décrits. L'urée, le cuivre et l'ergot ne sont pas cardiotoxiques.

4. **c)** The age of the calves, character of the diarrhea, lack of response to antimicrobial therapy, and finding of small oocysts all point to *C. parvum* which can cause diarrhea in humans via fecal-oral contact.

c) L'âge des veaux, le type de diarrhée, l'absence de réponse au traitement antimicrobien et la présence de petits oocystes, tout conduit à *C. parvum* qui peut causer de la diarrhée chez les humains par contact fécal-oral.

5. **c)** Clinical signs in ferrets with adrenocortical neoplasms include vulvar enlargement in females, squamous metaplasia of prostatic ducts and alveoli in males, bilaterally symmetric alopecia, polyuria, polydyspsia, anemia, and thrombocytopenia. The most consistent endocrinologic change in ferrets with adrenal neoplasms is elevated plasma concentration of estradiol-17 beta.

c) Les signes cliniques chez les furets souffrant de néoplasmes corticosurrénaliens comprennent un renflement de la vulve chez les femelles, de la métaplasie squameuse des canaux et des alvéoles prostatiques chez les mâles, de l'alopécie symétrique bilatérale, de la polyurie, de la polydipsie, de l'anémie et de la thrombocytopénie. Le changement endocrinologique le plus constant chez les furets souffrant de néoplasmes surrénaliens est une concentration plasmatique élevée d'estradiol-17 bêta.